(EEG).

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Eye Movement Disorders

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Oculogyric Crises

Oculogyric crises are a dystonia of ocular muscles[343] characterized by dramatic involuntary conjugate deviation of the eyes.[344] The eyes usually inadvertently move straight upward or up and to the left or right, and the position can change from crisis to crisis. Oculogyric crises are often associated with thought or emotional disturbances such as anxiety and restlessness, are preceded by a brief stare, and generally last from seconds to hours, with the entire episode lasting days to weeks.[345] Frequently they are accompanied by other dystonic or dyskinetic movements such as tongue protrusion, lip smacking, blepharospasm, choreoathetosis, anterocollis, and retrocollis.[346–348]

In the 1920s the French described crises (*crises oculogyres*) as complications of postencephalitic parkinsonism,[344,349] but today they are usually acute or tardive extrapyramidal reactions to neuroleptics (see Table 16.3), mainly first generation, but also second and third generation (aripiprazole,[350] quetiapine, [351] olanzapine,[352] ziprasidone[353]) In addition to postencephalitic parkinsonism, oculogyric crises have been associated with other neurologic disorders such as Parkinson's disease,[354] familial Parkinson's–dementia syndrome,[355] dopa-responsive dystonia,[356] parkinsonism with basal ganglia calcifications (Fahr disease),[357] neurosyphilis, multiple sclerosis, ataxia–telangiectasia,[358] Rett syndrome,[359] Wilson disease,[360] cerebellar disease, trauma,[361] acute herpetic brainstem encephalitis,[362] a third ventricular cystic glioma,[363] paraneoplastic disease,[339] midbrain lesions,[364] and striatocapsular infarction.[365,366] Besides neuroleptics, other medications have also been implicated (see Table 16.3).

The etiology of oculogyric crises is not certain. In postencephalitic patients, some authors believed they resulted from a release of supranuclear control of oculomotor centers as a result of injury to the corpus striatum or subthalamic nucleus.[344] Onuaguluchi[349] hypothesized they were due to an abnormal VOR in the setting of brainstem lesions involving vestibular pathways. Leigh et al.[345] attributed the deviations to an incorrect efference copy of eye position. Based on the response to anticholinergic agents in neuroleptic-induced crises, they alternatively invoked a defect in mesencephalic vertical gaze-holding mechanisms normally dependent on balanced cholinergic and dopaminergic systems.[345] Another hypothesis explains oculogyric crises as a limbic–motor disorder.[361]

Treatment. Severe or painful oculogyric crises can be treated acutely with benztropine or diphenhydramine, intramuscularly or intravenously.[361] A dose can be repeated in 30 minutes if there is no response. If these are unsuccessful, then diazepam or lorazepam, intramuscularly or intravenously, can be used. The underlying condition should be treated, or the offending drug removed or dosage lowered. Some patients with oculogyric crises due to neuroleptic use may require short-term treatment with oral benztropine or trihexyphenidyl for 2 weeks following the acute episode.[361] Seizures should be excluded with an electroencephalogram (EEG).

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